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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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LADAS & PARRY LLP
26 WEST 61ST STREET
NEW YORK, NY 10023

EXAMINER	
CHEN, SHIN LIN	

ART UNIT	PAPER NUMBER
1632	

NOTIFICATION DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

nyuspatactions@ladas.com

Office Action Summary	Application No.	Applicant(s)	
	10/556,640	PENG ET AL.	
	Examiner	Art Unit	
	Shin-Lin Chen	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-9* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-9* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *11-21-06 & 12-1-08* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *11-21-06, 2-22-08, 5-27-10*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's amendment filed 12-1-08 has been entered. Claims 1-7 have been amended. Claims 8 and 9 have been added. Claims 1-9 are pending and under consideration.

Specification

1. The amendment filed 12-1-08 is objected to under 35 U.S.C. 132(a) because it introduces new matter into the disclosure. 35 U.S.C. 132(a) states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the original disclosure is as follows: The added paragraphs on pages 1, 2, 4, 6 and 7 of the specification lack sufficient support in the originally filed specification. Applicant fails to point out where in the specification has the support for the added paragraphs in the amendment filed 12-1-08.

Applicant is required to cancel the new matter in the reply to this Office Action.

This application contains sequence disclosures that are encompassed by the definition for nucleotide and/or amino acid sequences set forth in 37 CFR 1.821 (a)(1) and (a)(2). However, this application fails to comply with the requirements of 37 CFR 1.821 through 1.825 because there is no sequence identifier for the nucleotide sequence on pages 2-5, 8, 10 and 12 of the specification and in Figure 3 or in the "BRIEF DESCRIPTION OF THE DRAWINGS". Each nucleotide sequence is required to have a sequence identifier. Appropriate correction is required.

Drawings

2. The drawings were received on 12-1-08. These drawings are accepted.

3. The drawings were received on 11-21-06. These drawings are accepted.

Claim Rejections - 35 USC § 112

4. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

5. Claims 1-9 are rejected under 35 U.S.C. 112, second paragraph, as being incomplete for omitting essential steps, such omission amounting to a gap between the steps. See MPEP § 2172.01. The omitted steps are: How the adenovirus vector is used to produce the medicine and whether the medicine is produced or not.

6. Claim 1 recites the limitation "the medicine" in the last line. There is insufficient antecedent basis for this limitation in the claim.

Claim Rejections - 35 USC § 112

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 1-9 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for using a recombinant adenovirus vector comprising a human p53 gene to inhibit tumor growth and to inhibit prostate cell growth in benign prostatic hyperplasia (BPH) via direct injection to the tumor and prostate, respectively, does not reasonably provide enablement

for a recombinant adenovirus vector comprising a p53 gene for treating various proliferative diseases, including tumors, via various administration routes in vivo. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

While determining whether a specification is enabling, one considered whether the claimed invention provides sufficient guidance to make and use the claimed invention, if not, whether an artisan would have required undue experimentation to make and use the claimed invention and whether working examples have been provided. When determining whether a specification meets the enablement requirement, some of the factors that need to be analyzed are: the breadth of the claims, the nature of the invention, the state of the prior art, the level of one of ordinary skill, the level of predictability in the art, the amount of direction provided by the inventor, the existence of working examples, and whether the quantity of any necessary experimentation to make or use the invention based on the content of the disclosure is "undue" (In re Wands, 858 F.2d at 737, 8 USPQ2d 1400, 1404 (Fed. Cir.1988)).

Furthermore, the USPTO does not have laboratory facilities to test if an invention with function as claimed when working examples are not disclosed in the specification, therefore, enablement issues are raises and discussed based on the state of knowledge pertinent to an art at the time of the invention, therefore skepticism raised in the enablement rejections are those raised in the art by artisans of expertise.

Claims 1-9 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA. Claim 3

specifies downstream of the gene expression cassette is any of the eukaryotic gene poly A tail. Claims 4 and 5 are directed to production of the recombinant gene medicine by using homologous recombination between pGT-1 plasmid and adenovirus in prokaryotic cells to produce pGT-2, and homologous recombination of pGT-2 and an artificial sequence containing the right arm of adenovirus/promoter-p53-poly A/ the left arm of adenovirus in prokaryotic cells, such as E. coli, wherein the prokaryotic sequence is discarded by suing endonuclease PacI. Claims 6-8 specify the proliferative disease is any kind of scar, pathological scar, or cheloid. Claim 9 specifies the recombinant is used to produce injection solution.

The specification discloses "the size of the scar had significantly decreased after gene therapy for 4 weeks" (page 14, Experiment 3). The specification fails to disclose what kind of "gene therapy" has been performed and how the therapeutic gene is administered in vivo. The claims encompass preparing a recombinant adenovirus vector containing a p53 gene for treating various proliferative diseases, including any kind of scar, pathological scar and cheloid as specified in claims 6-8, any disease that has overgrowth of cells and cancers etc., at different locations in a subject via various administration routes in vivo.

The claims read on gene therapy by using the claimed recombinant adenovirus vector in vivo. The state of the art for gene therapy was unpredictable at the time of the invention. While progress has been made in recent years for gene transfer *in vivo*, vector targeting to desired tissues *in vivo* continues to be unpredictable and inefficient as supported by numerous teachings available in the art. For example, Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicates that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long

enough period of time" (page 53, first paragraph). Verma et al., Sept. 1997 (Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). The claims encompass using various promoters for the expression of the p53 polypeptide at various target cells in a subject. Different promoters have different activity in stimulating gene expression in different cells in vivo and whether there is sufficient expression of the p53 polypeptide at target cells depends on what promoter is used.

Administration route also plays a very important role in determining whether sufficient p53 polypeptide can be expressed and present at the target cells at various locations in vivo. The administration route includes direct administration to the target cells, oral administration, intraperitoneal injection, topical administration, intravenous administration, intramuscular injection, and subcutaneous administration etc. There are various barriers before a nucleic acid construct can reach its target cells, for example, layers of dermal cells, blood vessel wall cell membranes, lysosomal degradation within cells, extracellular matrix between cells, and gastrointestinal digestive acids. Eck et al., 1996 (Goodman & Gilman's The Pharmacological Basis of Therapeutics, McGraw-Hill, New York, p. 77-101) reports that numerous factors complicate in vivo gene transfer with respect to predictably achieving levels and duration of gene expression which have not been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the

tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used, the protein being produced, and the disease being treated (e,g, bridging pages 81-82). Gorecki, 2001 (Expert Opin. Emerging Drugs, 6(2): 187-198) reports that "the choice of vectors and delivery routes depends on the nature of the target cells and the required levels and stability of expression" for gene therapy, and obstacles to gene therapy *in vivo* include "the development of effective clinical products" and "the low levels and stability of expression and immune responses to vectors and/or gene products" (e.g. abstract). In addition, Thomas et al., 2003 (Nature Reviews/ Genetics, Vol. 4, p. 346-358) discusses the problem of viral vector in gene therapy. Thomas reports that "adenovirus vectors induce multiple components of the immune response: cytotoxic T-lymphocyte (CTL) responses can be elicited against viral gene products or "foreign" transgene products that are expressed by transduced cells, and the capsid itself--- in the absence of viral gene expression --- induces humoral virus-neutralizing antibody responses and potent cytokine-mediated inflammatory responses (e.g. p. 352, right column). Absent specific guidance, one skilled in the art at the time of the invention would not know how to use the claimed recombinant adenovirus vector for treating various proliferative diseases, including any kind of scar, pathological scar and cheloid, any disease that has overgrowth of cells, and cancers etc., at different locations in a subject via various administration routes in vivo.

Further, different proliferative diseases differ physiologically and pathologically. The symptoms of various proliferative diseases differ from each other. The specification fails to provide adequate guidance and evidence for how to "treat" various proliferative diseases by using the claimed adenovirus vector in vivo. There is no evidence of record that demonstrate "treating" various proliferative diseases by using the claimed adenovirus vector such that the symptoms of the proliferative diseases are ameliorated via various administration routes in vivo.

For the reasons discussed above, it would have required undue experimentation for one skilled in the art at the time of the invention to practice over the full scope of the invention claimed. This is particularly true given the nature of the invention, the state of the prior art, the breadth of the claims, the amount of experimentation necessary, the level of skill which is high, the working examples provided and scarcity of guidance in the specification, and the unpredictable nature of the art.

Claim Rejections - 35 USC § 102

9. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.
> (e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

10. Claims 1, 2 and 9 are rejected under 35 U.S.C. 102(b) as being anticipated by Shirakawa et al., 2000 (The Journal of Gene Medicine, Vol. 2, p. 426-432, IDS).

Claims 1, 2 and 9 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA. Claim 9 specifies the recombinant is used to produce injection solution.

Shirakawa teaches preparation of Ad-CMV-p53, a replication-deficient recombinant adenovirus containing CMV promoter driving wt-p53 gene. The Ad-CMV-p53 was injected into rat ventral prostates for treating benign prostatic hyperplasia (BPH) and showed 30% decrease in average prostate weight (e.g. abstract). The Ad-CMV-p53 recombinant adenovirus contains CMV promoter, wt-p53 cDNA and SV40 polyadenylation signal in the expression cassette that replace the serotype 5 adenoviral E1 region (e.g. p. 427, left column, last paragraph). Since the Ad-CMV-p53 adenovirus was injected into rat ventral prostates, the adenovirus is in injection solution. Thus, the claims are anticipated by Shirakawa.

11. Claims 1 and 2 are rejected under 35 U.S.C. 102(e) as being anticipated by Higginbotham et al., 2005 (US Patent No. 6,875,610 B2).

Claims 1 and 2 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA.

Higginbotham teaches introduction of a therapeutic gene, such as p53 gene, into an adenoviral vector containing promoter-gene-poly A. The promoter can be any promoter known in the art and the poly A tail can be SV40 poly A tail (e.g. Brief Summary Text (13), Detailed Description Text (9)). Since p53 was known in the art as a tumor suppressor gene, therefore p53

is a therapeutic gene for treating cancer, which is a proliferative disease. Thus, the claims are anticipated by Higginbotham.

12. Claims 1, 2 and 9 are rejected under 35 U.S.C. 102(b) as being anticipated by Chiang et al., 2002 (US 20020051767 A1).

Claims 1, 2 and 9 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA. Claim 9 specifies the recombinant is used to produce injection solution.

Chiang teaches a process for improving the treatment of a tumor by radiation therapy comprising treating a tumor cell, transduced with an adenoviral vector containing a DNA sequence encoding wt-p53, with radiation. The adenoviral vector comprises an adenoviral 5' ITR, and adenoviral 3' ITR, an adenoviral encapsidation signal, a DNA sequence encoding wt-p53, and a promoter sequence (e.g. abstract, [0033]). An modified adenoviral vector can be constructed by constructing a shuttle plasmid containing an adenoviral 5' ITR, an adenoviral encapsidation signal, a multiple cloning site, a poly A signal, and a DNA segment which corresponds to a segment of the adenoviral genome, which serves as a substrate for homologous recombination with a modified or mutated adenovirus. The shuttle plasmid can also contain a selectable marker and an origin of replication from bacteria (e.g. [0037]). Since the adenoviral vector can be used to transduce tumor cells, the adenoviral vector is in injection solution. Thus, the claims are anticipated by Chiang.

Claim Rejections - 35 USC § 103

13. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

14. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

15. Claims 1-3 are rejected under 35 U.S.C. 103(a) as being unpatentable over Shirakawa et al., 2000 (The Journal of Gene Medicine, Vol. 2, p. 426-432, IDS) in view of Falck-Pedersen, E., 2004 (US Patent No. 6,824,770 B1).

Claims 1-3 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA. Claim 3 specifies downstream of the gene expression cassette is any of the eukaryotic gene poly A tail.

Shirakawa teaches preparation of Ad-CMV-p53, a replication-deficient recombinant adenovirus containing CMV promoter driving wt-p53 gene. The Ad-CMV-p53 was injected into rat ventral prostates for treating benign prostatic hyperplasia (BPH) and showed 30% decrease in

average prostate weight (e.g. abstract). The Ad-CMV-p53 recombinant adenovirus contains CMV promoter, wt-p53 cDNA and SV40 polyadenylation signal in the expression cassette that replace the serotype 5 adenoviral E1 region (e.g. p. 427, left column, last paragraph).

Shirakawa does not specifically teach using eukaryotic gene poly A tail in the expression cassette.

Falck-Pedersen teaches preparing a replication defective recombinant adenovirus, AdCNVCATgD, comprising CMV-1 promoter, bacterial CAT sequence and the mouse beta globin poly(A) site. The adenovirus sequence from 3.8-15.0 m.u. provides DNA sequence for homologous recombination (e.g. Detailed Description Text (97)).

It would have been prima facie obvious to one of ordinary skill in the art at the time of the invention to use an eukaryotic poly A tail in a recombinant adenovirus vector because Falck-Pedersen teaches using a mouse beta globin poly A site in a replication defective recombinant adenovirus.

One having ordinary skill in the art at the time the invention was made would have been motivated to do so in order to prepare a recombinant adenovirus containing p53 gene for treating benign prostatic hyperplasia (BPH) as taught by Shirakawa with reasonable expectation of success.

16. Claims 1, 4 and 5 are rejected under 35 U.S.C. 103(a) as being unpatentable over Chiang et al., 2002 (US 20020051767 A1) in view of He et al., 1998 (PNAS, Vol. 95, p. 2509-2514, IDS).

Claims 1, 4 and 5 are directed to an application of a recombinant of adenovirus vector and human tumor suppressor p53 gene expression cassette for producing the medicine for treating proliferative disease, wherein the recombinant composed of promoter-p53cDNA-polyA. Claims 4 and 5 are directed to production of the recombinant gene medicine by using homologous recombination between pGT-1 plasmid and adenovirus in prokaryotic cells to produce pGT-2, and homologous recombination of pGT-2 and an artificial sequence containing the right arm of adenovirus/promoter-p53-poly A/ the left arm of adenovirus in prokaryotic cells, such as E. coli, wherein the prokaryotic sequence is discarded by suing endonuclease PacI.

Chiang teaches a process for improving the treatment of a tumor by radiation therapy comprising treating a tumor cell, transduced with an adenoviral vector containing a DNA sequence encoding wt-p53, with radiation. The adenoviral vector comprises an adenoviral 5' ITR, and adenoviral 3' ITR, an adenoviral encapsidation signal, a DNA sequence encoding wt-p53, and a promoter sequence (e.g. abstract, [0033]). An modified adenoviral vector can be constructed by constructing a shuttle plasmid containing an adenoviral 5' ITR, an adenoviral encapsidation signal, a multiple cloning site, a poly A signal, and a DNA segment which corresponds to a segment of the adenoviral genome, which serves as a substrate for homologous recombination with a modified or mutated adenovirus. The shuttle plasmid can also contain a selectable marker and an origin of replication from bacteria (e.g. [0037]).

Chiang does not specifically teach homologous recombination between pGT-1 plasmid and adenovirus in prokaryotic cells, such as E. coli, to produce pGT-2, and the prokaryotic sequence of the recombinant adenovirus is discarded by suing endonuclease PacI.

He teaches advantages of homologous recombination in bacteria to generate recombinant adenoviruses over in eukaryotic cells (e.g. abstract). He teaches using adenoviral backbone vectors, pAdEasy-1 containing all Ad5 sequences except nucleotides 1-3,533 (encompassing E1 genes) and nucleotides 28,130-32,820 (encompassing E3) and shuttle vector containing adenoviral left arm and right arm (both contains LTR) for homologous recombination with pAdEasy-1 in E. coli. The shuttle vector and the pAdEasy-1 are mixed together to perform homologous recombination to produce resulting recombinant adenoviral vector DNA, which was digested with PacI for transfection of mammalian cells (e.g. p. 2510, left column, 2nd paragraphs to right column, 3rd paragraph).

It would have been prima facie obvious to one of ordinary skill in the art at the time of the invention to perform homologous recombination between pGT-1 plasmid and adenovirus in E. coli to produce pGT-2 vector because He teaches advantages of generating recombinant adenovirus in prokaryotic cells, such as E. coli, over in eukaryotic cells, and pGT-1 only needs to have ITR on both ends of adenovirus. The pAdEasy-1 as taught by He is an adenoviral backbone vector, which can be considered as pGT-2 that is formed by homologous recombination between a plasmid containing LTR on both ends and an adenovirus genome. It also would be obvious to one of ordinary skill in the art at the time of the invention to digest the recombinant adenovirus with endonuclease PacI because He teaches digesting the recombinant adenoviral vector DNA with PacI before transfection of mammalian cells.

One having ordinary skill in the art at the time the invention was made would have been motivated to do so in order to produce a recombinant adenoviral vector containing p53 gene for

treating a tumor as taught by Chiang or to generate a recombinant adenoviral vector via homologous recombination in E. coli as taught by He with reasonable expectation of success.

Information Disclosure Statement

17. The information disclosure statement (IDS) submitted on 11-21-06, 2-22-08 and 5-27-10 was filed before the mailing of the first Official action. The submission is in compliance with the provisions of 37 CFR 1.97. Accordingly, the information disclosure statement is being considered by the examiner. The foreign patent JP 2001-522871 has no translation, therefore, it is NOT considered by Examiner.

Conclusion

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Shin-Lin Chen whose telephone number is (571) 272-0726. The examiner can normally be reached on Monday to Friday from 9:30 am to 6 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras can be reached on (571) 272-4517. The fax phone number for this group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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Shin-Lin Chen, Ph.D.
/Shin-Lin Chen/
Primary Examiner
Art Unit 1632